How does an oscillatory drive shape the correlations in binary networks?

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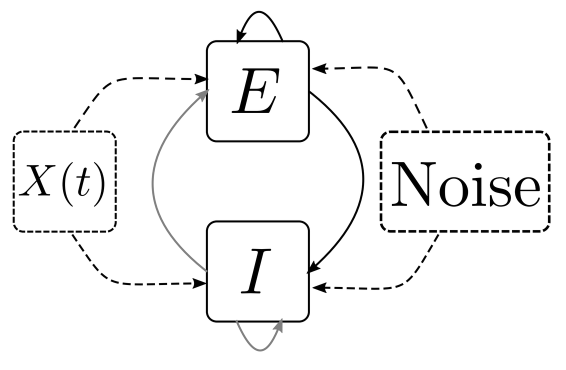
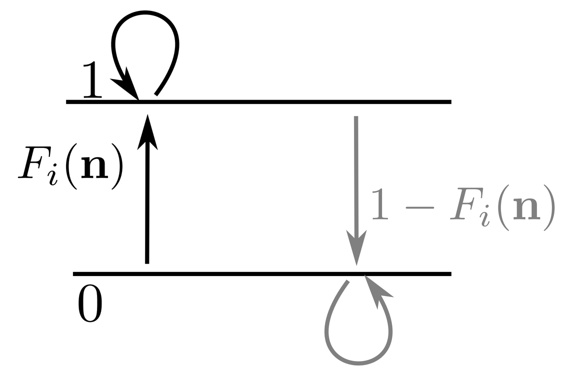
Binary neurons in Glauber dynamics

The theoretical framework and moments ODE



The mean activity of X modulates sinusoidally. The (uncorrelated Gaussian) noise is needed to smooth the nonlinear transfer function.



Update scheme for the Glauber dynamics: A single neuron i is chosen with the probability $\frac{dt}{\tau}$ for an update and ends in the up-state with probability $F_i(\boldsymbol{n})$, else in the down-state.

The model network is composed of binary neurons, which are described by a Master equation [4, 5]. As a gain function, we choose

$$F_i(\mathbf{n},t) = H(h_i(t)-\theta)\,,\ \text{where } h_i = \sum_{k=1}^{N} J_{ik} n_k(\omega t) + \xi_i\ ,\ H(x) = \begin{cases} 1 & \text{if } x \geq 0 \\ 0 & \text{if } x < 0 \end{cases} \tag{1}$$

J_{ij} is the synaptic weight for the connection $j \to i$ and ξ is uncorrelated Gaussian noise with ξ_i=0 and $\langle \xi_i \xi_j \rangle = \sigma^2_{\text{noise}}\delta_{ij}$ (equivalently, we could have chosen an error function instead of the Heaviside function and left out the Gaussian noise). The activity of the neurons in X is modulated in time such that on average, every neuron in the local population gets the input $h_{\text{ext}}\sin(\omega t)$ in addition compared to an external population with stationary activity [6].

We assume the network to be homogeneous, i.e. we set $J_{ij} = J_{\alpha\beta}\ \forall i,j \in \alpha,\beta$, where α and β are one of the three populations (exc., inh. and extern) and we assume a fixed number of connections $K_{\alpha\beta}$ between α and β. Due to the homogeneity of the network setting, we set $m_\alpha := \langle n_i \rangle\ \forall i \in \alpha$ and $h_\alpha := \langle h_i \rangle\ \forall i \in \alpha$.

We use a Gaussian closure to solve the hierarchy of moment equations, where h_α is a Gaussian random variable determined by the parameters μ_α and σ_α given by

$$\begin{aligned}\mu_\alpha(t) &= \sum_\beta K_{\alpha\beta}J_{\alpha\beta}m_\beta + h_{\text{ext}}\sin(\omega t)\\ \sigma^2_\alpha &= \sum_{\beta,\beta'=1}^{\tilde{N}} K_{\alpha\beta}K_{\alpha\beta'}J_{\alpha\beta}J_{\alpha\beta'}c_{\beta,\beta'} + \sum_{\beta=1}^{\tilde{N}} K_{\alpha\beta}J^2_{\alpha\beta}\left(m_\beta - m_\beta^2\right) + \sigma^2_{\text{noise}}.\end{aligned} \tag{2}$$

Mean activities

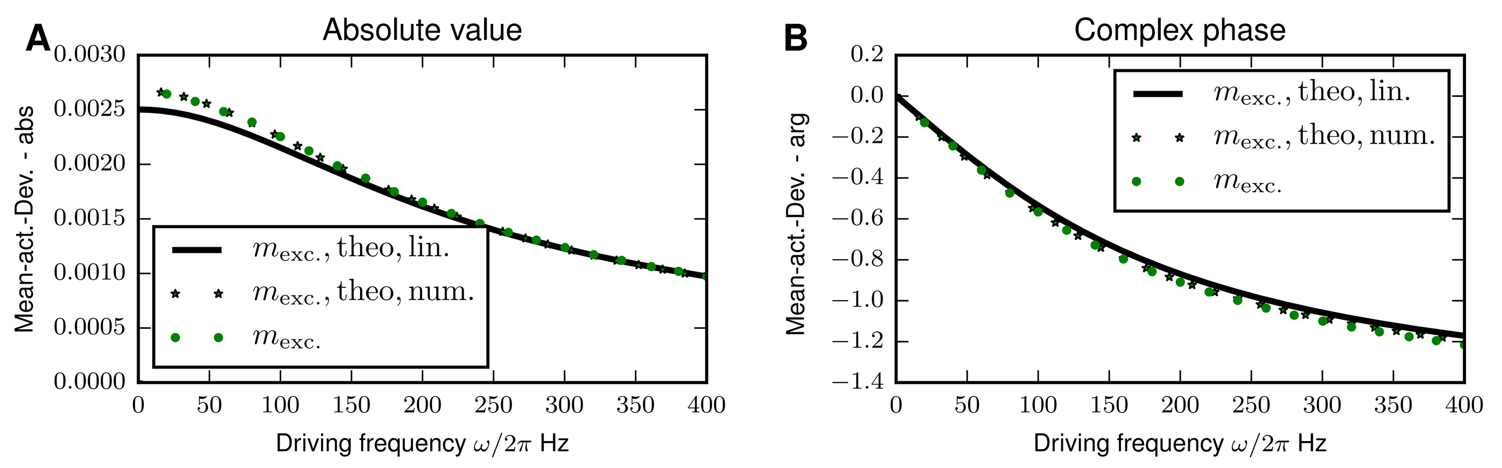
From the Master equation, we derive an ODE for the population averaged mean activity m_α

$$\tau\frac{d}{dt}m_\alpha(t) = -m_\alpha(t) + \langle H(x_\alpha(t)-\theta)\rangle = -m_\alpha(t) + \frac{1}{\sqrt{\pi}}\int_{\frac{\theta-\mu_\alpha(t)}{\sqrt{2}\sigma_\alpha}}^{\infty} e^{-x^2}\,dx. \tag{3}$$

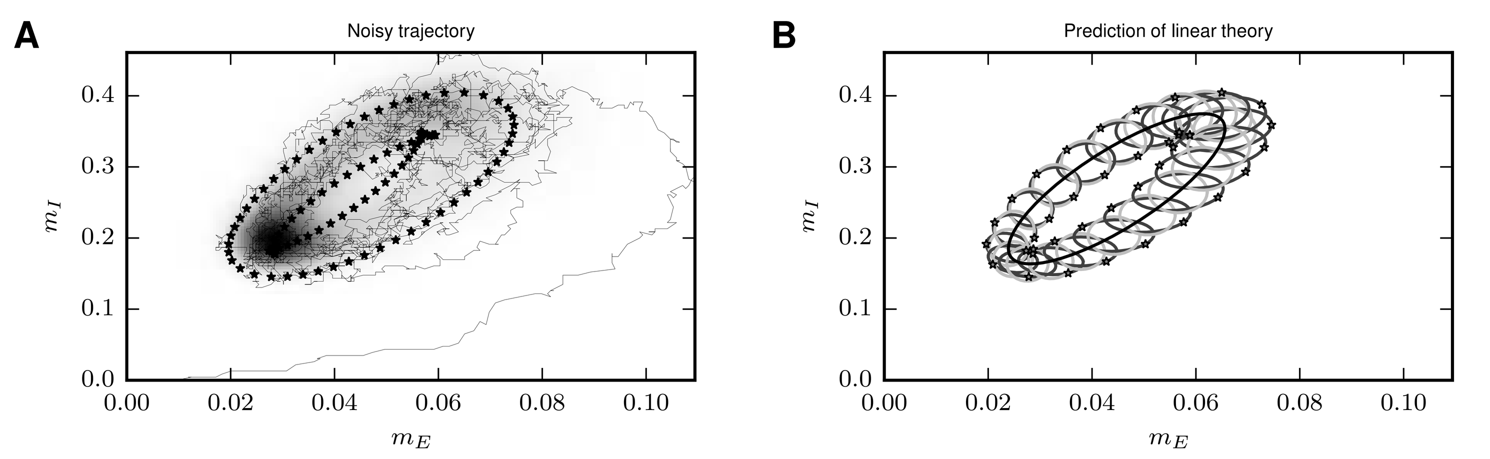
Setting the time derivative to zero and solving (numerically) for m_α gives the stationary mean activity $\overline{m}_\alpha$. Small deviations $\delta m_\alpha(t)$ thereof are calculated by expanding the rhs of eq. (3) in $\delta\mu_\alpha(\delta m_\alpha) := \mu_\alpha(\delta m) - \mu^0_\alpha \approx \frac{d\mu_\alpha}{dm_\alpha}\delta m_\alpha$ and h_{ext} (we neglect the contribution from $\delta\sigma_\alpha$). That gives

$$\tau\frac{\partial}{\partial t}\delta m_\alpha(t) + \delta m_\alpha(t) = \overline{S}_\alpha\left\{\left[(K\circ J)\,\boldsymbol{\delta m}(t)\right]_\alpha + h_{\text{ext}}\sin(\omega t)\right\},\ \text{where } \overline{S}_\alpha := \frac{1}{\sqrt{2\pi}\overline{\sigma}_\alpha}e^{-\frac{(\overline{\mu}_\alpha-\Theta_\alpha)^2}{2(\overline{\sigma}_\alpha)^2}}. \tag{4}$$

The ansatz $\delta m_\alpha(t) = \text{Im}\left(M_\alpha e^{i\omega t}\right)$ gives $M_\alpha = h_{\text{ext}}\sum_\beta U_{\alpha\beta}\frac{\left(U^{-1}S\right)_\beta(-i\tau\omega+1-\lambda_\beta)}{(\tau\omega)^2+(1-\lambda_\beta)^2}$.



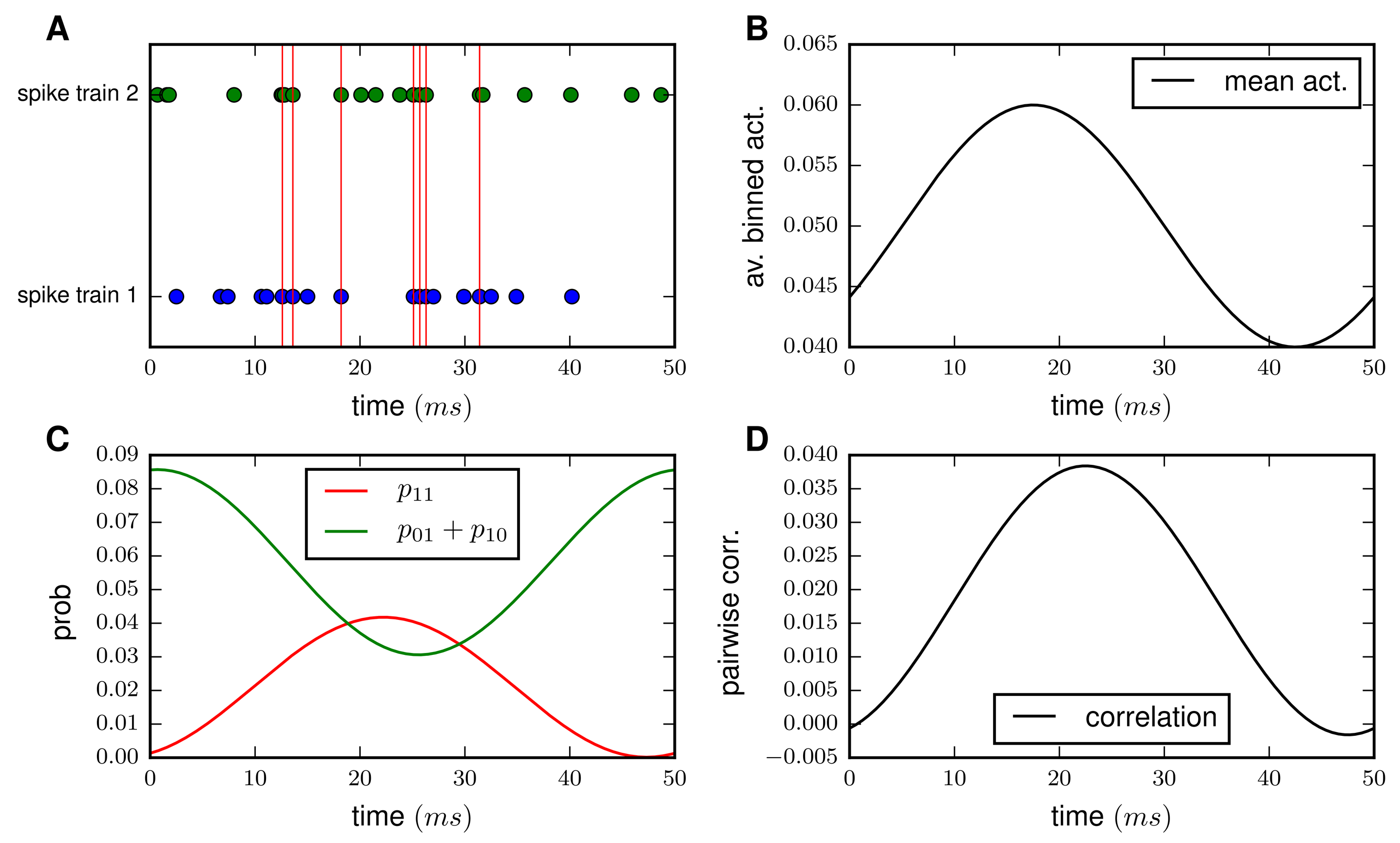
Absolute value (A) and complex phase (B) of $\delta m_{exc.}$. Parameters as in parts of [5]: $N_E = N_I = N_X = N = 8192$, $j_{\alpha X} = j_{\alpha E} = \frac{5}{\sqrt{N}}$, $p_{\alpha\beta} = p = 0.2\ \forall\alpha,\beta$, $j_{\alpha E} = \frac{-10}{\sqrt{N}}$, $\tau = 10ms$, $m_E = m_I \approx 0.11$, $m_X = 0.25$, noise level $\sigma^2 = pNj^2_{\alpha X}\left[0.5^2 - m_X(1-m_X)\right]$.



Panel A: Probability for simulated network in a activity state (m_E, m_I) denoted in gray shades. Grey line: Sample trajectory of the binary system. Dots: Prediction of the linear theory for the region of maximally one standard deviation distance from the limit cycle of the mean activity. Their construction is shown in panel B: Draw error ellipse (given by inverse of correlation matrix) around a point $\left(m^0_E, m^0_I\right)$ on the limit cycle. The stars (corresponding to the dots in A) marks the points on the error ellipse with the largest distant to the tangent of the limit cycle at $\left(m^0_E, m^0_I\right)$. Parameters: $N_E = 1691$, $N_I = 230$, $N_X = 500$, $p_{EE} = 0.168$, $p_{IE} = 0.327$, $p_{EI} = 0.5$, $p_{II} = 0.36$, $p_{EX} = 0.2$, $p_{IX} = 0.3$, $j_{EE} = j_{EX} = 0.37$, $j_{IE} = j_{IX} = 0.82$, $j_{EI} = -0.52$, $j_{II} = -0.54$, $m_E = 0.045$, $m_I = 0.27$, $m_X = 0.1$, $\tau = 2.5ms$, $\frac{\omega}{2\pi} = 20Hz$, $\boldsymbol{\sigma}_{\text{noise}} \approx 5\boldsymbol{\sigma}_{\text{loc. network}}$.

Experimental motivation

Locking of spikes to LFP and cell assemblies



The correlation of spiking activity is locked to certain phases of the (periodic) local field potential (LFP).

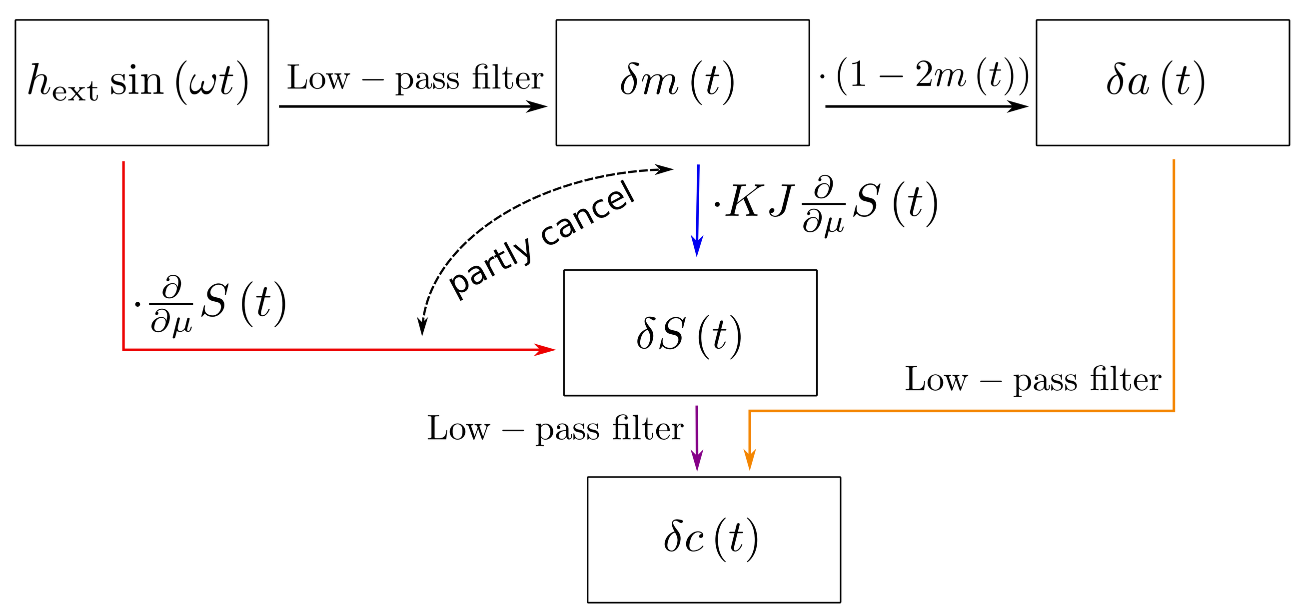
- Occurence of simultaneous spikes exceeding the level expected for independently active neurons is more strongly locked to the LFP cycle than neurons firing simultaneously coincidentally [3] → Cell assemblies?
- Excess spiking synchrony in experiments quantified by unitary event (UE) analysis ([1], [2]), can be related to pairwise correlations.
- LFP often considered to primarily reflect input to the local population. → Add as strongly simplified LFP-related periodic input to the local model neurons a sinusoidal modulation.
- Assumption for our model: "Null hypothesis", i.e. there are no cell assemblies and all neurons receive the external input in the same way.

Results: Linear approximation yields good prediction for first two moments, **"resonance"** in frequency-dependent correlations mainly resulting from the **interplay** of the modulation of the susceptibility produced by **external drive** and by the **modulation of the mean activity**.

Pairwise correlations

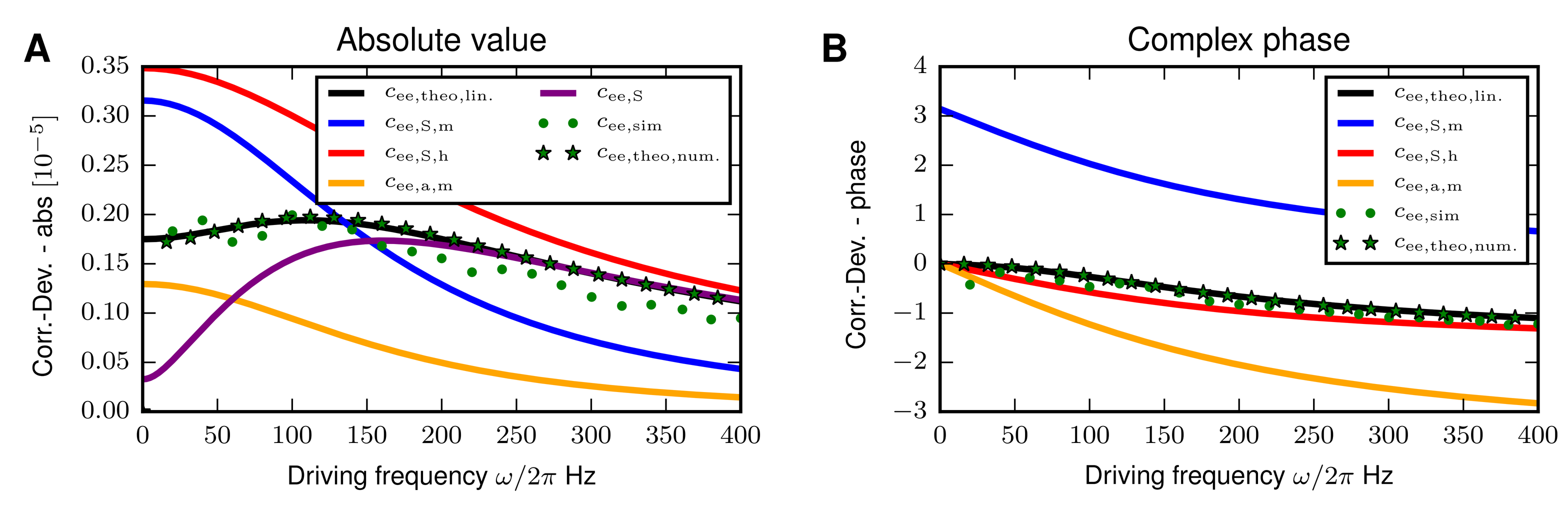
Invoking again the Master equation, we get after neglecting all cumulants of order higher than 2 (Gaussian closure) the differential equation for $c_{\alpha\beta}(t) = \frac{1}{N_\alpha N_\beta}\sum_{i\in\alpha,j\in\beta} c_{ij}(t)$:

$$\tau\frac{d}{dt}c_{\alpha\beta}(t) + 2\cdot c_{\alpha\beta}(t) = \left\{S(\mu_\alpha(t),\sigma_\alpha)\left[(K\circ J)\left(c(t) + \text{diag}\left(\frac{a_\beta(t)}{N_\beta}\right)\right)\right]_{\alpha\beta}\right\} + \{\alpha \leftrightarrow \beta\} \tag{5}$$



Linear approximation in h_{ext} and δm of susceptibilities and autocorrelations gives the drive for δc.

- Expand eq. (5) about the stationary values $\overline{c}_{\alpha\beta}$, $\overline{m}_\alpha$ to linear order in h_{ext}, $\delta m(t)$ and $\delta c(t)$ to get an ODE for $\delta c(t)$.
- Contribution coming from modulated autocorrelations is low-pass-filtered twice, therefore decays like $\frac{1}{\omega^2}$ asymptotically.
- Contributions from the modulated susceptibility come from the direct drive (asymptotically like $\frac{1}{\omega}$) and the recurrent feedback (asymptotically like $\frac{1}{\omega^2}$). In addition, they have opposite signs → "Resonance".



The absolute values (A) and phases (B) of the first harmonic of the pairwise correlation in linear and non-linear mean field theory from the

References

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